CLINICAL AND BIOCHEMICAL PROFILE OF WOMEN PRESENTING WITH HIRSUTISM AND IT'S TREATMENT OUTCOME-A PROSPECTIVE STUDY

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BRANCH II

INSTITUTE OF OBSTETRICS AND GYNAECOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI – 600003.

APRIL 2012

CERTIFICATE

This is to certify that the dissertation entitled **"CLINICAL AND BIOCHEMICAL PROFILE OF WOMEN PRESENTING WITH HIRSUTISM AND ITS TREATMENT OUTCOME-A PROSPECTIVE STUDY"** done by Dr. D.S.BEBINCY, to the faculty of Obstetrics and Gynaecology, the Tamil Nadu Dr.M.G.R.Medical University, Chennai in partial fulfilment for the award of M.D.Degree (Obstetrics and Gynaecology) is a bonafide research work carried out by her under our direct supervision and guidance.

Dr.V.KANAGASABAI M.D.
Dean,
Madras Medical College,
Chennai - 600003.

Dr.M.MOHANAMBAL MD,DGO
Director and Superintendent
Institute of Obstetrics &
Gynaecology
Madras Medical College
Chennai – 600003.

DECLARATION

I hereby declare that the study entitled **CLINICAL AND BIOCHEMICAL PROFILE OF WOMEN PRESENTING WITH HIRSUTISM AND IT'S TREATMENT OUTCOME-A PROSPECTIVE STUDY** was done by me in the Institute of Obstetrics and Gynaecology (IOG), Madras Medical College, Chennai – 600003, during the period of my PG study for MD Branch II Obstetrics and Gynaecology from 2010 – 2011.

This Dissertation to Dr.M.G.R. Medical University is in partial fulfilment of University regulations for the award of MD Degree in Obstetrics and Gynaecology.

Dr.D.S.Bebincy
M.D. PG (Obstetrics and Gynaecology)
Institute of Obstetrics and Gynaecology
Madras Medical College,Chennai-600 003.

Place :

Date :

I have no objection to guide this postgraduate student in the project mentioned above. I shall supervise to the extent that all the human rights are protected and research is carried on with utmost humanitarian principles.

SIGNATURE OF THE GUIDE **SEAL OF THE GUIDE.**

INSTITUTIONAL ETHICAL COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. D.S. Bebincy
PG in MDOG
Institute of Obstetrics & Gynaecology
Madras Medical College, Chennai -3.

Dear Dr. D.S. Bebincy

The Institutional Ethical Committee of Madras Medical College reviewed and discussed your application for approval of the project / proposal / clinical trail entitled " Clinical and biochemical profile of hirsuitism" No 31082010.

The following members of Ethical committee were present in the meeting held on 17.08.2010 conducted at Madras Medical College,Chennai -3

1. Prof. S.K. Rajan, MD	-- Chairperson
2. Prof. J. Mohanasundaram, MD,Ph.D,DNB Dean, Madras Medical College, Chennai -3	-- Deputy Chairman
3. Prof. A. Sundaram, MD Vice Principal , MMC, Chennai -3	-- Member Secretary
4. Prof R. Nandhini, MD Director, Institute of Pharmacology, MMC, Ch-3	-- Member
5. Prof. Pregna B. Dolia , MD Director, Institute of Biochemistry, MMC, Ch-3	-- Member
6. Prof. C. Rajendran , MD Director, Institute of Internal Medicine, MMC, Ch-3	-- Member -- Member
7. Prof. V. Shruti Kamal, MS Professor of Surgery, MMC, Ch-3	-- Member
8. Mrs. Arnold Sauliina, Social Scientist	-- Member

We approve the trail to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee

ACKNOWLEDGEMENT

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CONTENTS

INTRODUCTION

INTRODUCTION

Hirsutism is a disorder which affects about 10 to 15 percent of women. Hirsutism is the most frequent androgen excess disorder in women[1]. Hirsutism is defined as the excessive growth of terminal hair in androgen dependent areas of a woman's body. The sites affected include

- The upper lip
- Chin
- Chest
- Upper abdomen
- Lower abdomen
- Upper back
- Lower back
- Upper Arm
- Thighs

Each of the nine areas is given a score of 0 to 4 depending upon the grade 1 – mild, 2-moderate, 3-complete light coverage and 4 for complete heavy coverage. A score of more than 15 is considered as severe hirsutism[2]. Occasionally, hirsutism may signal more serious pathology. Therefore, clinical evaluation should differentiate benign causes from tumours and other conditions that require specific treatment.

PICTURE NO.1

PICTURE OF A SUBJECT WITH EXCESSIVE HAIR IN THE ABDOMEN

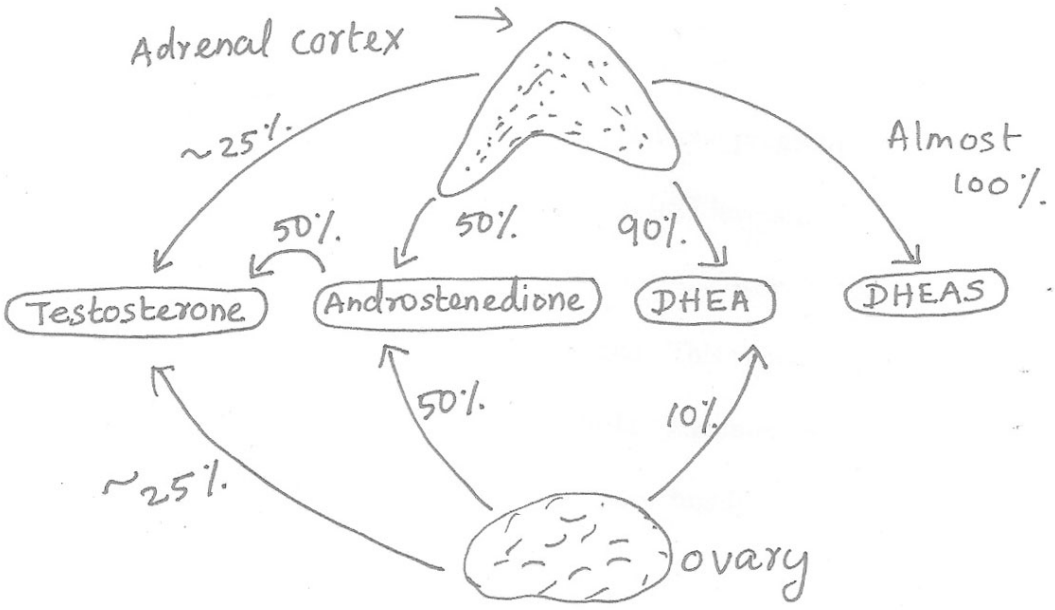
Hirsutism should be differentiated from **hypertrichosis.** Hypertrichosis is defined as a diffuse increase in vellus hair growth and is not androgen dependent. The congenital causes of hypertrichosis include Hurlers syndrome, trisomy 18, fetal alcohol syndrome etc. The other causes include hypothyroidism, porphyrias, epidermolysis bullosa, anorexia nervosa, malnutrition, dermatomyositis, severe head injury and trauma.[3].

Drugs causing hypertrichosis[4]

- Cyclosporin (Sandimmune)
- Diazoxide (Hyperstat)
- Hydrocortisone
- Minoxidil (Rogaine)
- Pencillamine (Cuprimine)
- Phenytoin (Dilantin)
- Psoralens (Oxsoralen)
- Streptomycin

PICTURE NO.2

SOURCES OF ANDROGENS IN ADULT WOMEN



Approximately one-half of a woman's serum testosterone is derived from peripheral conversion of secreted androstenedione, and the other-half is derived from direct glandular secretion. The ovaries and adrenal glands contribute almost equally to the direct glandular testosterone production in women[1].

PATHOGENESIS OF HIRSUTISM

Hirsutism can result from an increase in androgen level or due to the oversensitivity of the hair follicles to androgen. Large quantities of circulating androgens are bound to sex hormone binding globulin (SHBG), cortisol- binding globulin and albumin. The free testosterone is the main bioactive component of plasma testosterone. The SHBG can decrease in the body in many conditions such as obesity, hyperinsulinemia or after administration of androgens, synthetic progestins, glucocorticoids and growth hormones. With the reduction in the SHBG levels in the body, the free testosterone level increases which can result in hirsutism. However the severity of hirsutism does not correlate well with the level of the androgen. This is because the sensitivity of hair follicles to androgen varies among individuals. The enzyme 5 alpha-reductase type 1 converts testosterone to the highly active dihydrotestosterone .[5]

In an update on hirsutism, it was stated that familial clustering of PCOS, hyperandrogenism and hirsutism suggest a genetic etiology. Environmental factors might also be the triggering agents in some families. Recent experimental evidence supports the hypothesis of intrauterine environment influencing hyperandrogenic phenotype in adult life.[5]

PICTURE NO.3

PICTURE OF A SUBJECT WITH EXCESSIVE FACIAL HAIR

Both insulin and LH stimulate ovarian theca cell androgen production. As a result, affected ovaries secrete elevated levels of testosterone and androstenedione[6].

Insulin resistance independent of obesity has also been described as pathognomonic of PCOS[7]. Clinically, PCOS is characterized by menstrual irregularities, hyperandrogenism, hyperinsulinemia and long term metabolic disturbances such as diabetes mellitus, cardiovascular disease and dyslipidemias[8]. Insulin and body fat play an important role in regulating lipid levels[9].

PICTURE NO.4

PICTURE OF A SUBJECT WITH EXCESSIVE CHEST HAIR

Although both testosterone and DHT convert short, soft vellus hair to coarse terminal hair, DHT is markedly more effective than testosterone. Conversion is irreversible and hence only hairs in androgen sensitive areas are changed in this manner to terminal hairs[6].

CAUSES OF HIRSUTISM

Hirsutism is one sign of hyperandrogenism. Hirsutism can be familial, idiopathic, due to excess androgen secretion by the ovary and adrenal glands or from exogenous sources of androgens such as medications.

- Idiopathic hirsutism is often familial. The onset occurs shortly after puberty with slow progression. They have normal menses and normal androgen levels.[3]
- Ovarian causes-PCOS, androgen secreting ovarian tumors, menopause. The cardinal features of PCOS are hyperandrogenism and polycystic ovaries.[3]
- Adrenal causes-tumours, CAH, Cushing's syndrome
- Drug induced hirsutism is due to the following drugs.
    - Anabolic steroids
    - Metoclopramide
    - Phenothiazines
    - Progestins
    - Reserpine
    - Testosterone

Other causes include severe insulin resistance, anorexia nervosa, hyperprolactinemia, acromegaly, hypothyroidism and porphyria. Androgen secreting tumours of the ovary or adrenal gland usually present with virilization. Arrhenoblastoma, sertoli leydig cell tumour, theca cell tumour and some cases of granulosa cell tumour cause virilization.

Signs of virilization are

- Acne
- Seborrhea
- Clitoromegaly
- Deepening of voice
- Increased libido
- Increased muscle mass
- Infrequent or absent menses
- Loss of breast tissue or normal female contour
- Temporal hair recession and balding.

PICTURE NO.5

PICTURE OF A SUBJECT WITH CLITOROMEGALY

(This subject presented with amenorrhea and virilization. She underwent TAH with BSO. She was diagnosed as having Sertoli Leydig cell tumour).

Androgen secreting tumours of the ovary or adrenal are usually heralded by virilization, rapid progression of hirsutism and cessation of menses. Androgen secreting adrenal tumours are less common[10].

PICTURE NO.6

PICTURE OF A SUBJECT WITH FRONTAL HAIR LOSS

(She was diagnosed as having malignant granulosa cell tumour)

Thin skin striae or bruising are signs of hypercortisolism. Although hirsutism is the most common complaint associated with hyperandrogenism, signs of virilization reflect higher serum androgen levels.

Although rare, Cushing's syndrome should be considered in the differential diagnosis. It may be due to increased production of adrenocorticotropic hormone by the pituitary, adrenal carcinoma/adenoma or secretion of adrenocorticotropic hormone. Profound hirsutism is seen most commonly in patients with macronodular hyperplasia[3].

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Male-like hair growth, masculinization of women and ambiguity of gender has fascinated mankind for millennia, frequently appearing in mythology and the arts. The earliest reports of androgen excess, beginning 400 years BC focused on the appearance of male like hair growth and features in women often accompanied by menstrual cessation. The first of the etiologies identified were adrenal disorders, primarily adrenocortical neoplasms and adrenal hyperplasia.

Kozloviene et al ., at Lithuania studied clinical and hormonal changes in women aged 18 – 35 years who complained of hirsutism and concluded that significantly higher levels of testosterone and dehydroepiandrosterone sulfate, higher levels of free androgen index and lower levels of sex hormone binding globulin ($p<0.01$) were found in females with hirsutism. The females with hirsutism complained more frequently of infertility, increased greasiness of skin, had higher body mass index, higher systolic and diastolic blood pressure, larger waist and hip circumference and higher WHR.[11]

Escobar and San Mill et al., concluded that basal serum 17(OH) progesterone measurement has an excellent diagnostic performance for congenital adrenal hyperplasia and CAH needs to be excluded in hyperandrogenic women.[12]

Carmina and Rosato et al ., concluded that classic PCOS is the most common androgen excess disorder and mild androgen excess disorders like ovulatory PCOS and idiopathic hyperandrogenism are also common.[13]

In a study by Azziz et al., on 873 patients, the prevalence of androgen secreting neoplasms was 0.2%, 21 hydroxylase deficient classic CAH was 0.6% , 21 hydroxylase deficient nonclassic CAH was 1.6%, hyperandrogenic insulin resistant acanthosis nigricans (HAIRAN) syndrome was 3.1%, idiopathic hirsutism was 4.7% and PCOS was 82% . The study showed that hirsutism, menstrual dysfunction or acne improved in the majority of patients treated with a combination suppressive therapy but alopecia did not improve on treatment.[14]

Pekhlivanov et al., concluded that in women with expressed hirsutism, it is more appropriate to apply metformin as a monotherapy or in combination with programs for body weight reduction. Hirsutism improved when metformin was given.[15]

Carmina et al., concluded that the addition of dexamethasone to antiandrogen therapy for hirsutism prolonged the duration of remission.[16]

In a study conducted by Marescalchi et al ., at Institute of Obstetrics & Gynaecology, University of Bologna, Italy, it was concluded that despite different effects on androgen levels, flutamide, finasteride and EE-CPA constitute very satisfactory alternative therapeutic regimens in the treatment of hirsutism.[17]

Seaman and De Vries et al ., concluded that the risk of venous thromboembolism associated with EE-CPA does not differ significantly from that associated with the use of conventional COCs[18].

Cochrane data base analysis showed that OCP containing EE-CPA resulted in subjective improvement in hirsutism.[19]

Fleming et al ., compared the efficacy of metformin with OCP containing cyproterone acetate in the management of hirsutism[20].

Falsetti et al ., showed the effect of long term treatment (60 cycles) with the EE-CPA Pill and the follow up after 6 months from cessation in PCOS. Mild to moderate hirsutism disappeared in 36 – 60 cycles, where as severe hirsutism decreased substantially but persisted after 6 months from the end of the therapy. Endocrine parameters were identical to the starting ones. Acne and hirsutism reappeared whereas ovarian morphology was in between the initial and final condition.[21]

Rautio et al., at Finland studied the effects of metformin and EE-CPA on lipid levels in obese and nonobese women with PCOS. HDL increased, and serum triglyceride levels decreased during metformin therapy. In the OCP group, total cholesterol increased, HDL increased, total cholesterol/HDL decreased and TGL and LDL remained unchanged. Both systolic and diastolic pressures decreased over 6 months of metformin treatment. It had been shown that EE-CPA with metformin has been shown to improve the lipid profile and insulin sensitivity in nonobese women with PCOS.[22]

Aesha sadaf Hameed et al., made a comparative analysis between ethinyl estradiol with CPA with or without metformin in the treatment of PCOS induced hirsutism. Metformin when added to OCP containing CPA has a better outcome in treating hirsutism, menstrual irregularity and BMI reduction[23].

Ancuta Gheorghisan – Geluteanu reported a Sertoli Leydig cell tumour in postmenopausal women with hirsutism. There was marked reduction in hirsutism after resection of tumour.[10]

V ATAY, C CAM, M MUHCU et al ., 2006 concluded that letrozole is associated with better ovulation induction 16.5% and higher pregnancy rate and as a first line treatment for anovulatory patients with PCOS.[24]

In a study conducted by 2 clinics in Berlin and Hamburg in 170 Oligomenorrheic patients, hyperandrogenemia (increased testosterone and / or DHEAS) was seen in 41.8%, hyperprolactinemia in 25.9%, abnormal thyroid function (TSH and / or TRH induced TSH) in 21.7% and hypergonadotropic FSH levels in 3.5% of all patients[25].

AIM OF THE STUDY

AIM OF THE STUDY

There are not many studies that look at the causes of hirsutism, their clinical profile and hormonal changes .Therefore, this study was undertaken to analyse the clinical profile, biochemical profile, various causes of hirsutism and the response to treatment.

MATERIALS AND METHODS

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Study design

This is a prospective study.

Period of study

The period of study was from August 2010 to September 2011

Place of study

Subjects were taken from Endocrinology clinic and Fertility Research centre, Institute of Obstetrics and Gynaecology, Govt Hospital for Women and Children, Egmore.

Inclusion Criteria

Females with modified Ferriman-Gallwey scoring >8 were included in the study.

After getting an informed consent from the subjects, they were subjected to clinical and biochemical evaluation after history taking.

Exclusion criteria

Women who were 60 years and above and pregnant women were excluded from the study

History

History of presenting complaints were enquired

Age of onset of hirsutism and duration were asked. This is because severe hirsutism can occur in a short duration in androgen secreting tumours. Longer duration of hirsutism is present in idiopathic hirsutism.

Menstrual history: Any histories of irregular cycles or amenorrhoea were asked for. Menstrual complaints are common in PCOS, hypothyroidism, CAH and androgen secreting tumours. Amenorrhea is more common in CAH and androgen secreting tumours.

Duration of infertility: Infertility is common in PCOS. Elevated androgen levels prevent ovulation leading to infertility.

Recent weight gain: Weight gain occurs in PCOS, hypothyroidism, insulin resistance. Peripheral fat favours the conversion of testosterone to dihydrotestosterone. Weight loss is seen in malignant tumours.

History of drug intake:

Several drugs cause hirsutism. Name of the drug taken and the duration of the drug intake were also enlisted.

Clinical examination

The patients in the study were subjected to clinical evaluation.

Hirsutism scoring was made according to modified Ferriman-Gallwey scoring system[26].

Height in cm and weight in kg were measured.

Blood pressure was recorded in the sitting position in the right arm.

Body Mass index was calculated by the formula, weight in kg/(height in metre)^2

BMI (kg/m^2)	**weight status**
<18.5	Under weight
18.5-24.9	Normal
25.0-29.9	Overweight
30.0-39.9	Obese
40.0 and more	Extreme obesity

Waist circumference (in cm) and hip circumference (cm) were measured for all subjects. Waist was measured at the level of belly button and hip was measured at the broadest level of the gluteal region. Waist hip ratio was calculated for all subjects. Normal WHR is 0.8. WHR > 0.8 is considered as a marker of insulin resistance.

The subjects were examined for signs of virilization and hyperandrogenism like acne, male pattern balding, increased muscle mass, deep voice, clitoromegaly, breast atrophy. Acanthosis nigricans was also looked for in each subject. Features of Cushing's syndrome like moon facies and buffalo hump

were also looked for. Features of hyperprolactinemia like milk secretion were also looked for.

USG Pelvis

USG evidence of PCOS: Subjects who had 12 or more follicles in each ovary with size 2-9 mm and ovarian volume >10ml in one or both ovaries are considered as having polycystic ovaries[27]. Adnexal masses if any or any uterine abnormalities were also looked for.

USG Abdomen

USG abdomen was taken in cases suspected of congenital adrenal hyperplasia or ovarian mass in addition to USG pelvis.

CT Abdomen

CT abdomen was taken in cases of congenital adrenal hyperplasia and malignant ovarian tumours.

Biochemical Investigations

Free testosterone and dehydroepiandrosterone sulphate levels from venous blood were measured in all individuals. Free testosterone is a more sensitive indicator because it is the unbound hormone which causes hirsutism. Venous blood for free testosterone was taken from the subjects in the morning. DHEAS is

a direct measure of adrenal androgen activity. Hence it was also measured in all subjects. 17 (OH) progesterone level was measured in cases suspected of CAH and adrenal tumours[32]. TSH is also measured in all cases since hypothyroidism also causes hirsutism. 24hrs urinary cortisol has to be measured in cases suspected of Cushing's syndrome. Fasting blood sugar was measured from the venous blood for all subjects. Fasting blood sugar up to 125 mg/dl was considered as normal value and values between 126-200 mg/dl was considered as impaired glucose tolerance[34]. Values >200 mg/dl are considered as DM because insulin resistance can also cause hirsutism .100g OGTT is done in cases with fasting blood sugar > 125 mg/dl. PCOS is also associated with insulin resistance. Lipid profile was taken in all the subjects after 10 hrs fasting in the morning to rule out hyperlipidemia. These investigations were done to identify any co morbid illnesses like Diabetes mellitus and hyperlipidemia and to treat them at an early stage to prevent long term complications like atherosclerosis, stroke etc. Also PCOS is associated with metabolic syndrome.[28]

STANDARD LAB VALUES

- Free testosterone 0.4 - 2.0 pg/ml
- DHEAS 0.8-10.5 ng/ml
- TSH-0.5 to 5.5 mU/ml
- Lipid profile
- Total cholesterol <200 mg/dl

TGL < 150 mg/dl

HDL>55 mg/dl

LDL<130 mg/dl

Lipid profile and TSH are measured in the morning in empty stomach by enzymatic method.

Treatment

Subjects with idiopathic hirsutism and PCOS were given oral contraceptive pills containing cyproterone acetate 2mg with ethinyl estradiol 35mcg for 6months (6 cycles) and the outcome evaluated. The outcome evaluated were reduction in severity of hirsutism, regularization of cycles. In those with weight gain, metformin is added in a dosage of 500mg BD daily and looked for reduction in weight as well. Previous studies in women with documented PCOS have indicated that weight loss reduces insulin resistance and hyperandrogenism.[28,29,30]

In those with infertility, OCP containing cyproterone acetate 2mg and 35mcg ethinyl estradiol was given for 3 months along with metformin if BMI > 25 kg/m^2. Then letrozole was given for 3 cycles in a dosage of 2.5 mg OD for 5 days starting from the second day of the cycles. Metformin was also continued. Follicular study is done starting from 9^{th} day of cycles and subjects were advised intercourse when the follicle reaches 18-20 mm. Subjects were asked to report to the hospital if there was a missed period or onset of menses. Pregnancy was confirmed by urine gravindex test and USG pelvis in the first trimester at 8 weeks.

In those with ovarian tumours, definitive surgery was done depending on the stage and followed by chemotherapy if needed. For late onset congenital adrenal hyperplasia, dexamethasone 0.5 mg OD is given and looked for response to treatment. This is given to suppress the excess androgen levels. They also show features of virilization. They may need procedures like clitoroplasty also.

Local therapies

For patients with mild hirsutism, local measures such as shaving, bleaching and depilatories may suffice. Shaving is the easiest and safest method. Bleaching products are often ineffective for dark hair growth and skin irritation may occur. Electrolysis is one of the most and permanent methods of hair removal and may be an adjunct to hormonal treatment[3].

PHARMACOLOGY OF DRUGS

Pharmacology of drugs used

Cyproterone acetate combined with ethinyl estradiol acts as a contraceptive agent. It is given for 21 days with a gap of 7 days in a cycle.

Cyproterone acetate has progestational activity which inhibits LH release augmenting the direct antiandrogenic action. It competes with dihydrotestosterone for the intra cellular androgen receptor and inhibits its binding.

In case of PCOD, it increases SHBG which binds the free testosterone thereby reducing hair growth, acne & dry skin and regularizes the menstrual cycle.

Side Effects:

- Prevent pubertal changes (larger doses)
- Loss of libido (adults)
- Gynecomastia (males)
- Amenorrhea (large doses)

Uses:

- Contraception ,Acne
- Virilization (women)
- Regularization of cycles

Metformin: It is a biguanide. It cause little or no hypoglycemia in non diabetic individuals and even in diabetics, hypoglycemia is rare. In addition to its action on blood sugar, it improves lipid profile in type 2 diabetes.

Mechanism of action:

- Suppresses hepatic gluconeogenesis and glucose output from liver.
- Enhance insulin – mediated glucose disposal in muscle & fat through the transport of GLUT1 from intraceullular site to plasma membrane.
- Promote peripheral glucose utilization by enhancing anaerobic glycolysis.

Pharmacokinetics: Excreted unchanged by kidney.

Adverse effects:

- Abdominal pain
- Anorexia
- Nausea
- Metallic taste
- Mild diarrhoea
- Tiredness

Letrozole:

It is an aromatase inhibitor, a orally active nonsteroidal compound. It reversibly inhibits aromatization all over the body, resulting in total oestrogen deprivation. It has 100% oral bioavailability.

Use: Ovulation induction.

Advantages: Causes unifollicualar development. Has no effect on cervical mucus.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

TEST STATISTICS USED WERE

- Chi – square test
- two sample ‘t’ test
- paired ‘t’ test

The subjects after clinical, biochemical analysis and ultrasonogram examination were grouped into PCOD and non PCOD. The treatment was given according to the cause of hirsutism. The outcome evaluated were weight reduction, regularity of menstrual cycles and reduction in the scoring of hirsutism. After treatment, 5 from the PCOD group and 3 from the non PCOD group conceived. There were 6 defaulters in the study. Two postmenopausal subjects were advised LASER treatment. They resorted to shaving the facial hair. One subject with malignant sertoli leydig cell tumor had recurrence of the tumour and died due to metastasis. A p value<0.05 is significant

TABLE NO.1

AGE GROUP – GROUP STATISTICS

Group	**No**	**Mean**	**Standard deviation**	**Standard error mean**
PCOD	46	24.11	4.175	0.616
NON PCOD	27	30.15	9.172	1.765

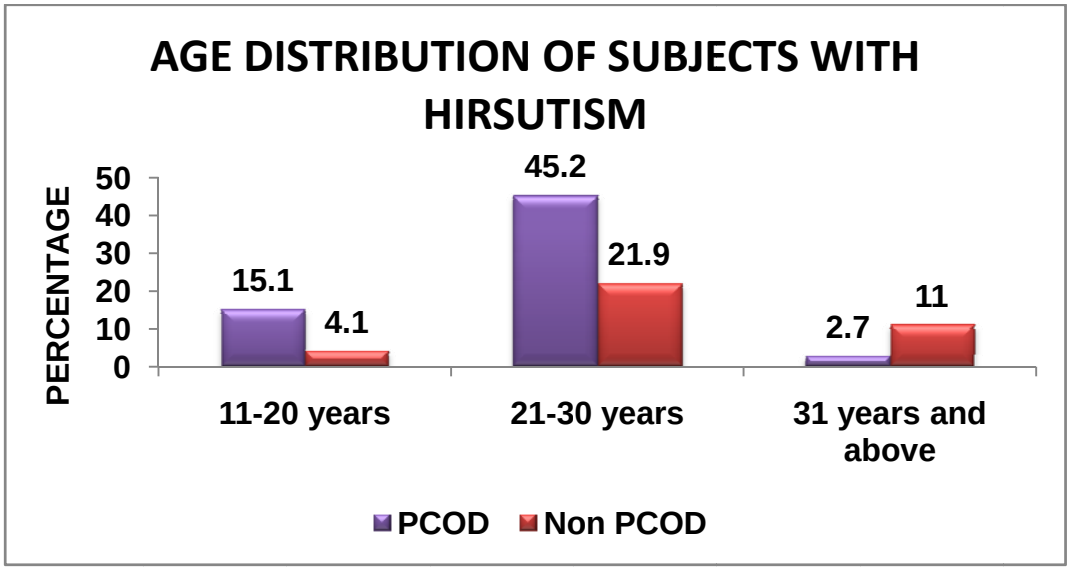
The mean age in the PCOD group is 24.11 years and in the non PCOD group, it is 30.15 years (p value < 0.05). It is statistically significant.

TABLE NO.2

AGE GROUP – CROSS TABULATION

Age group	PCOD	NON PCOD	TOTAL
11 – 20 yrs % of total	11 15.1%	3 4.1%	14 19.2%
21 – 30 yrs % of total	33 45.2%	16 21.9%	49 67.1%
31 years and above % of total	2 2.7%	8 11%	10 13.7%
Total % of total	46 63%	27 37%	73 100%

CHART NO.1



CHI- SQUARE TESTS

	Value	df	Asymp. Sig (2sided)
Pearson Chi-Square	9.787[a]	2	.007
Likelihood Ratio	9.735	2	.008
Linear-by-Linear Association	7.471	1	.006
N of Valid Cases	73		

a. 1 cells (16.7%) have expected count less than 5. The minimum expected count is 3.70.

We infer that there exists a statistical significance between PCOD and non PCOD group with reference to age distribution

TABLE NO.3

HISTORY OF SUBJECTS WITH HIRSUTISM

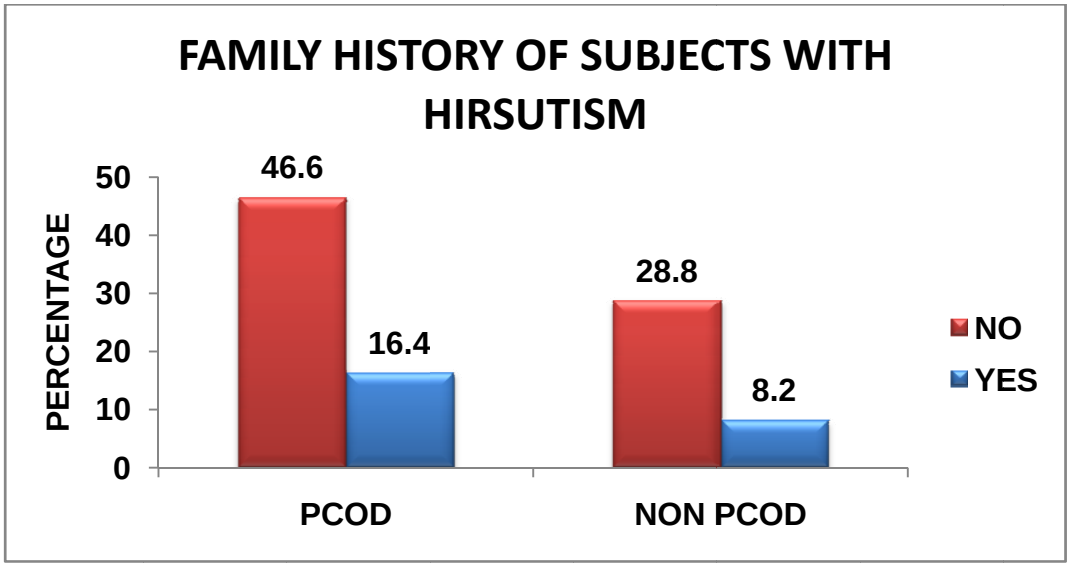
	PCOD	**NON PCOD**	**Significance**
Marital status Married unmarried	 25(34.2%) 21(28.8%)	 12 (16.4%) 15(20.5%)	p =0.414
Family history of hirsutism	12(16.4%)	6(8.2%)	p= 0.711
Drug intake	0	2	p=0.0296

TABLE NO.4

FAMILY HISTORY OF SUBJECTS WITH HIRSUTISM

Family History	PCOD	NON PCOD
NO % within group % of Total	34 73.9% 46.6%	21 77.8% 28.8%
Yes % within group % of Total	12 26.1% 16.4%`	6 22.2% 8.2%
Total	46	27

CHART NO.2



There is no statistical significance between PCOD and NON PCOD group with reference to marital history, family history and drug intake in this study. There is a statistical significance between both the groups with regard to drug intake. (p=0.0296)

TABLE NO.5

PRIMARY PRESENTING COMPLAINTS OF SUBJECTS WITH HIRSUTISM.

	PCOD	NON PCOD	Total	Singificance
Infertility	22(30.1%)	5(6.8%)	27(37%)	P=0.012
Menstrual irregularities	36(49.3%)	15(20.5%)	51(69.9%)	P=0.041
Weight gain	44(60.3%)	19(26.0%)	63(86.3%)	P=0.002

There exists a statistical difference between PCOD and non PCOD groups with respect to the complaints of infertility, menstrual irregularities and weight gain.

CHART NO.3

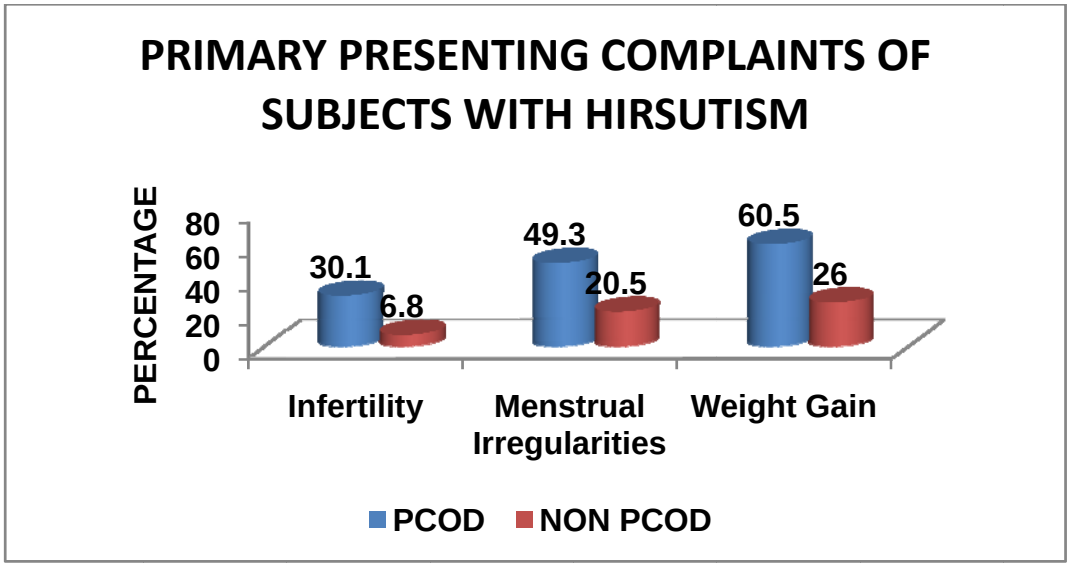


TABLE NO.6

DURATION OF HIRSUTISM OF SUBJECTS IN THE STUDY

GROUP STATISTICS

GROUPS	Total NO	Mean	Standard Deviation	Standard error mean
PCOD	46	4.89	3.622	0.534
NON PCOD	27	5.52	5.767	1.110

The mean duration of hirsutism in the PCOD group is 4.89 years and in the non PCOD, it is 5.52 years. It is statistically insignificant. (p=.569)

INDEPENDENT SAMPLES TEST

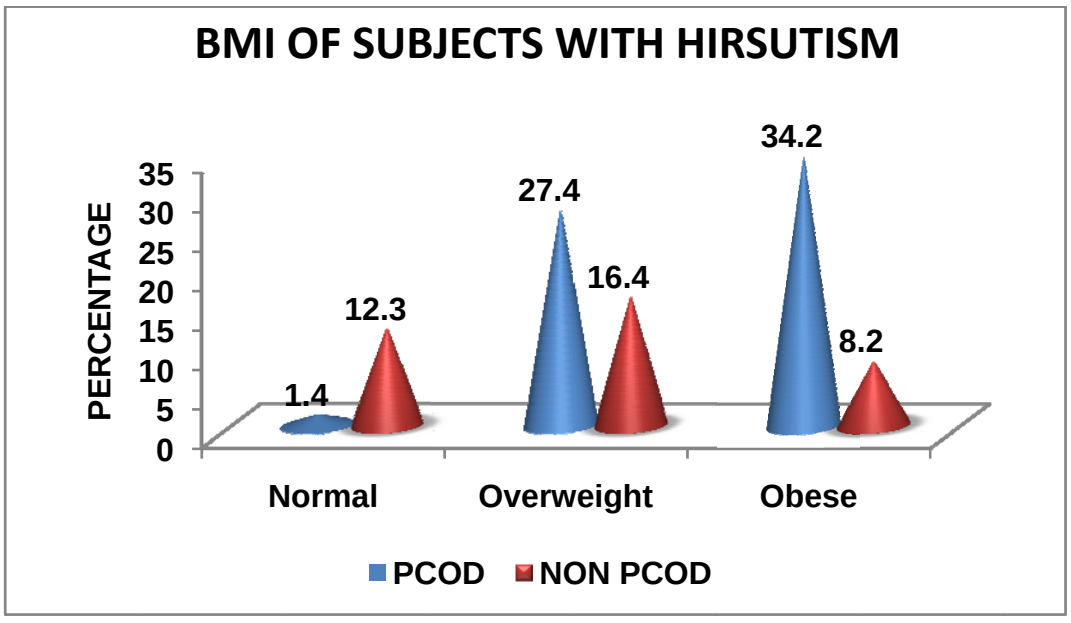
Duration of hirsutism	Levene's Test for Equality of Variances		t-test for Equality of Means		
	F	Sig.	t	df	Sig (2-tailed)
Equal variances assumed Equal variances not assumed	5.306	.024	-.571 -.509	71 38.250	.569

TABLE NO.7

BMI OF SUBJECTS WITH HIRSUTISM IN THIS STUDY

BMI	PCOD	NON PCOD	Total
18.5-24.9 % of total	1(1.4%)	9(12.3%)	10(13.7%)
25-29.9 % of total	20(27.4%)	12(16.4%)	32(43.8%)
>= 30 % of total	25(34.2%)	6(8.2%)	31(42.5%)

CHART NO.4



CHI-SQUARE TESTS

	Value	df	Asym.sig (2-sided)
Pearson Chi-Square	16.197[a]	2	0.000
Likelihood Ratio	16.893	2	0.000
Linear-by-Linear Association	14.034	1	0.000
N of Valid Cases	73		

a. 1 Cells (16.7%) have expected count less than 5. The minimum expected count is 3.70

p=0.000

There exists a statistical significance in the BMI of subjects between the PCOD and the non PCOD group.

TABLE NO.8

BMI – GROUP STATISTICS

	Total No:	Mean	Standard Deviation	Standard Error Mean
PCOD	46	29.36	2.813	0.415
NON PCOD	27	26.39	4.261	0.820

p=0.001

TABLE NO.9

GROUP STATISTICS

WAIST HIP RATIO OF SUBJECTS WITH HIRSUTISM

	Total No:	**Mean**	**Standard Deviation**	**Standard Error Mean**
PCOD	46	0.83	0.20	0.003
NON PCOD	27	0.82	0.033	0.006

Here p value is 0.095

Mean waist hip ratio of subjects with PCOD and hirsutism is 0.83 and in the subjects in the non PCOD group, the mean waist hip ratio is 0.82. It is statistically insignificant.

INDEPENDENT SAMPLES TEST

Waist hip ratio	**Levene's Test for Equality of Variances**		**t-test for Equality of Means**		
	F	Sig.	t	df	Sig. (2-tailed)
Equal variances assumed	10.170	.002	1.694	71	.095
Equal variances not assumed			1.500	37.598	

TABLE NO.10

BIOCHEMICAL PARAMETERS OF SUBJECTS WITH HIRSUTISM

	PCOD	NON PCOD	Total	Significance
Hyperlipidemia **% within group** **% of total**	5 10.9% 6.8%	7 25.9% 9.6%	12 16.4% 16.4%	0.094
Elevated free testosterone **% within group** **% of total**	17 37% 23.3%	7 25.9% 9.6%	24 32.9% 32.9%	0.333
Elevated DHEAS **% within group** **% of total**	6 13% 8.2%	9 33.3% 12.3%	15 20.5% 20.5%	0.038
Elevated TSH **% with group** **% of total**	2 4.3 2.7	6 22.2% 8.2%	8 11.0% 11.0%	0.018

CHART NO.5

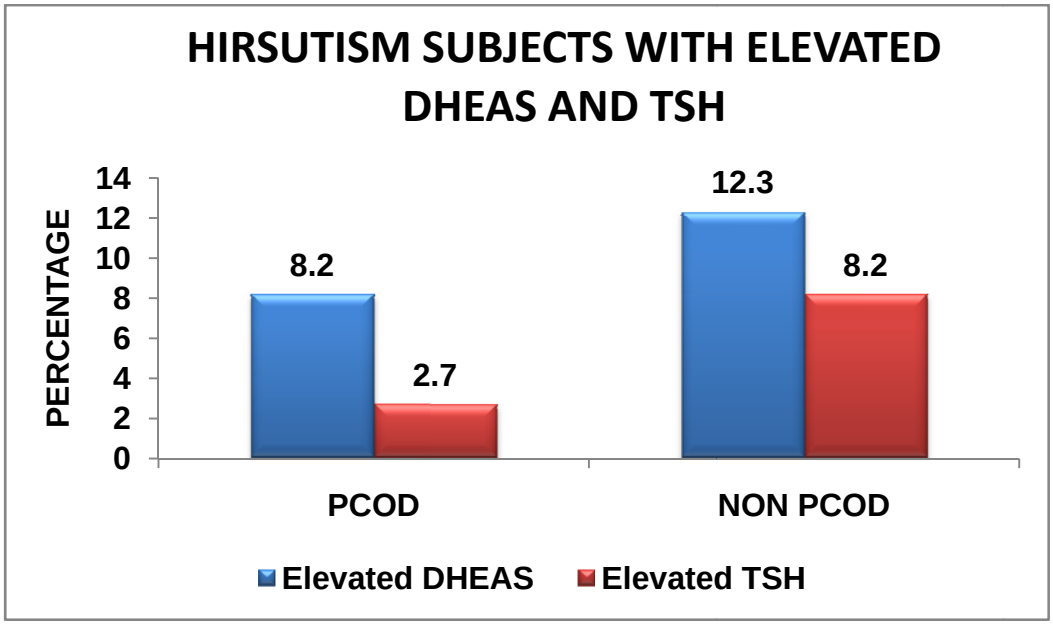


TABLE NO.11

HIRSUTISM SUBJECTS WITH NORMAL AND ELEVATED FREE TESTOSTERONE LEVELS

FREE TESTOSTERONE	PCOD	NON PCOD
Normal **% within group** **% of total**	29 63% 39.7%	20 74.1% 27.4%
Elevated **% within group** **% of total**	17 37% 23.3%	7 25.9% 9.6%
Total	46	27

CHART NO.6

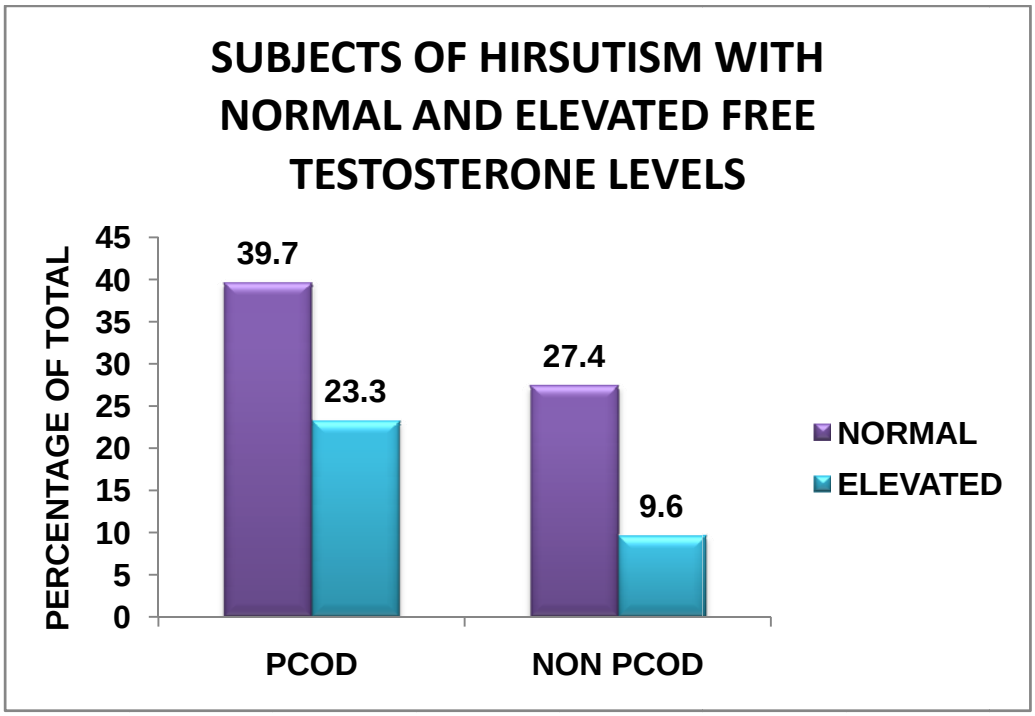
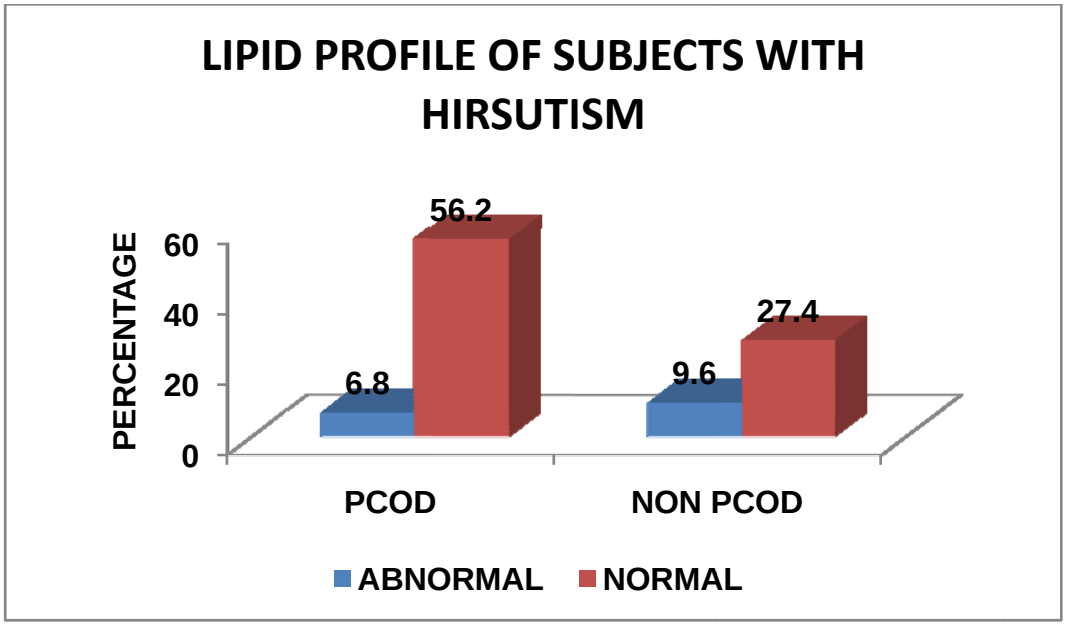


TABLE NO.12

LIPID PROFILE OF SUBJECTS WITH HIRSUTISM

Lipid profile	PCOD	Non PCOD
Abnormal **% within group** **% of total**	5 10.9% 6.8%	7 25.9% 9.6%
Normal **% within group** **% of total**	41 89.1% 56.2%	20 74.1% 27.4%
Total count	46	27

CHART NO.7



There exists a statistical significance in the elevated TSH levels and elevated DHEAS levels between the PCOD and the non PCOD groups. There is no statistical significance between the PCOD and non PCOD groups in elevated free testosterone levels and hyperlipidemia.

TABLE NO.13

GROUP STATISTICS- FASTING BLOOD SUGAR

	Total No:	**Mean**	**Standard Deviation**	**Standard Error Mean**
PCOD	46	88.98	15.937	2.350
NON PCOD	27	98.04	26.690	5.136

Here p value = 0.073

It is statistically not significant.

TABLE NO.14

GROUP STATISTICS – FREE TESTOSTERONE LEVELS

	Total No:	**Mean**	**Standard Deviation**	**Standard Error Mean**
PCOD	46	1.92	1.164	0.172
NON PCOD	27	2.18	2.048	0.394

Here p value = 0.494. The mean free testosterone level in the PCOD group is 1.92 pg/ml and in the non PCOD group it is 2.18 pg/ml.

TABLE NO.15

GROUP STATISTICS – DHEAS LEVELS

	Total No:	**Mean**	**Standard Deviation**	**Standard Error Mean**
PCOD	46	8.64	1.941	0.286
NON PCOD	27	8.76	2.114	0.407

Here p value = 0.806

TREATMENT OUTCOME OF SUBJECTS WITH HIRSUTISM

TABLE NO.16

PRE TREATMENT HIRSUTISM SCORING OF SUBJECTS WITH HIRSUTISM

		Total No:	**Mean**	**Standard Deviation**	**Standard Error Mean**	**Significant**
Pre treatment hirsutism	**PCOD**	46	11.61	2.695	0.397	p=0.798
	NON PCOD	27	11.44	2.517	0.484	

Mean hirsutism score is 11.61 in the PCOD group and 11.44 in the non PCOD group. It is statistically insignificant.

TABLE NO.17

REDUCTION OF HIRSUTISM SCORING AFTER TREATMENT IN THE PCOD GROUP

Paired Samples Statistics

PCOD Group	Mean	No.	Standard deviation	Standard error
Hirsutism score Pre treatment	11.69	42	2.789	.430
Hirsutism Post treatment	10.69	42	3.272	.505

p=0.000

There is a statistically significant reduction in hirsutism score after treatment in the PCOD group. The mean reduction is 1.

TABLE NO.18

REDUCTION IN HIRSUTISM SCORING AFTER TREATMENT IN THE NON PCOD GROUP

Non PCOD Group	Mean	No.	Standard deviation	Standard error
Hirsutism score Pre treatment	11.59	22	2.462	0.525
Hirsutism Post treatment	10.41	22	2.282	0.486

p=0.001

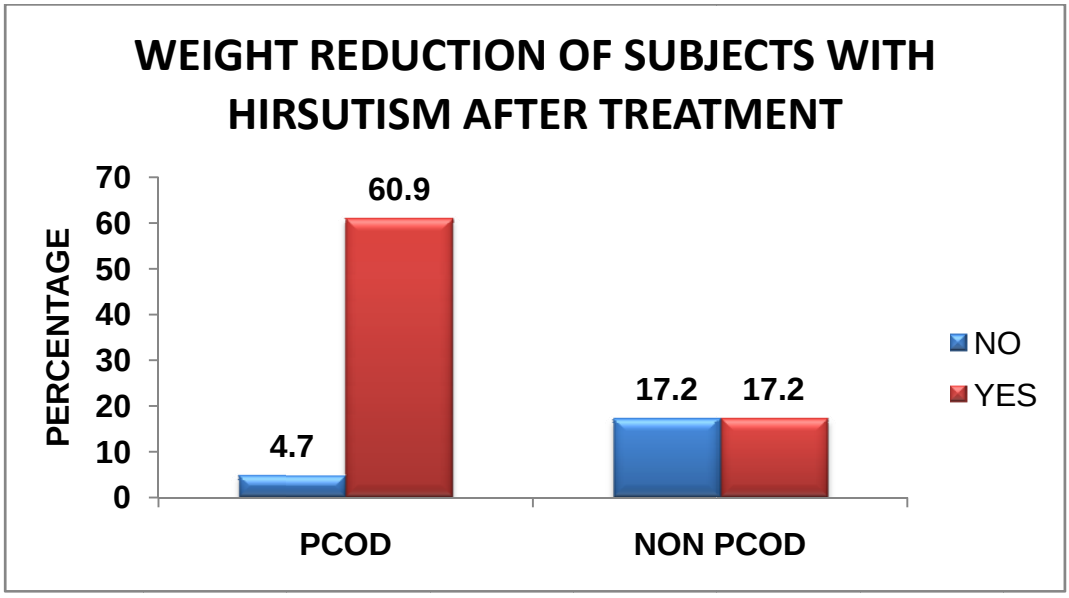
There exists a statistical significance between the hirsutism scores of the pre treatment and post treatment Non PCOD subjects. These is a mean reduction in score by 1.18

TABLE NO.19

WEIGHT REDUCTION IN THE HIRSUTISM SUBJECTS AFTER TREATMENT

Weight Reduction	PCOD	Non PCOD	Total
NO % of total	3 (4.7%)	11 (17.2%)	14 (21.9)
Yes % of total	39 (60.9%)	11 (17.2%)	50 (78.1%)

CHART NO.8



Chi-Square tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-square	15.517[a]	1	.000		
Continuity correction[b]	13.110	1	.000		
Likelihood Ratio					
Fisher's Exact Test	15.128	1	.000		
N of Valid Cases				.000	.000
	64				

a. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 4.81.

b. Computed only for a 2x2 table

p = 0.000

There is a statistically significant reduction in weight after treatment in hirsutism subjects in this study.

TABLE NO.20

OUTCOME OF THE MENSTRUAL CYCLES IN HIRSUTISM SUBJECTS AFTER TREATMENT OF HIRSUTISM

	PCOD	Non PCOD	Total
Regular % of total	37 57.8%	16 25%	53 82.8%
Irregular cycles % of total	2 3.1%	1 1.6%	3 4.7%
Oligomenorrhea or Amenorrhea % of total	3 4.7%	3 4.7%	6 9.4%
Premature menopause or Surgical menopause % of total	0	2 3.1%	2 3.1%

CHI-SQUARE TESTS

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.255[a]	3	.354
Likelihood Ratio	3.078	3	.380
Linear-by-Linear Association	3.040	1	.081
N of Valid Cases	64		

a. 6 cells (75.0%) have expected count less than 5. The minimum expected count is 1.03. Here p=.081. It is statistically insignificant.

USG pictures of subjects with hirsutism

- Polycystic ovaries – 46.
- Hypoplastic uterus with adrenal hyperplasia-1
- Adnexal mass – 6.
    - **Dermoid cyst – 1**
    - **Endometriotic cyst-1**
    - **Ovarian mass-4**
- Normal Study – 21

DISCUSSION

DISCUSSION

A study by Aesha Sadaf et al., at Bahawalpur, Pakistan showed the age range of hirsutism was from 16 to 38 years. In our study the age range of hirsutism was from 15 to 51 years[23]. In our study 65% of PCOS subjects had onset of hirsutism between 10 and 20 years whereas 50% of non PCOS subjects had onset of hirsutism from 21 and 30 years.

In a study on 122 female residents by Kozloviene at al., at Lithuania aged 18-35 years in 2002-2003 with complaints of hirsutism, hirsutism was present in 74 (60.66%) females and in 48 females (39.34%) no hirsutism was noted. The females with hirsutism complained more frequently of infertility ($P<0.05$), increased greasiness of skin ($p<0.05$), higher systolic and diastolic blood pressure, larger waist and hip ratio ($P<0.001$), higher levels of testosterone and dehydroepiandrosterone sulphate ($P<0.05$)[11]. In our study, the subjects complained of infertility ($p=.012$), irregular cycles (n=31) % oligomenorrhoea or amenorrhea (n=12). Increased waist hip ratio was seen in 14 subjects, increased free testosterone in 24 cases and increased DHEAS in 15 subjects($p=.038$) in our study. Two subjects with hirsutism had premature ovarian failure . They had increased FSH and LH. They were given hormone replacement therapy.

In a study by Carmina et al., at Palermo, Italy between 1980 and 2004, the prevalence of androgen excess disorders was PCOS (72.1%), idiopathic hyperandrogenism (15.8%), idiopathic hirsutism (7.6%), 21-hydroxylase – deficient non classic congenital adrenal hyperplasia (4.3%), androgen secreting

tumours (0.2%)[13]. In our study conducted between August 2010 and September 2011, the prevalence of PCOS was 63.01%, idiopathic hirsutism 0.44%, hypothyroidism 0.08% androgen secreting ovarian tumours 0.05%, premature ovarian failure (0.02%), drug induced hirsutism 0.02%, late onset congenital adrenal hyperplasia 0.014%[12]. A 15 years old girl who presented with features of virilization including hirsutism was diagnosed as congenital adrenal hyperplasia-late onset type.

In the study by Azziz et al., at Alabama between October 1987 and June 2002 in 873 subjects, the prevalence of androgen secreting neoplasm was 0.2%, 21-hydroxylase-deficient classic adrenal hyperplasia was 0.6% 21-hydroxylase – deficient nonclassic adrenal hyperplasia was 1.6%, hyperandrogenic insulin – resistant acanthosis nigricans (HAIRAN) syndrome was 3.1%, idiopathic hirsutism was 4.7% and PCOS was 82%. Fifty-nine patients (6.75%) had elevated androgen levels, hirsutism and normal ovulation. A total of 257 patients were included in the assessment of the response to hormone therapy. The mean duration of follow up was 33.5 months (range 6-15.5). Hirsutism improved in 86%, menstrual dysfunction in 80%, and hair loss in 33% of patients[14].

In our study, free testosterone levels were elevated in 24 subjects (24/73=32.87%). DHEAS in 14/73=19.17%), abnormal lipid profile in 12/78=16.4%), elevated TSH in 8 subjects (10.95%). The response was assessed in 64 subjects after 6 months of treatment. Subjects who were post menopausal (n=2) were advised laser treatment. They did not go for laser treatment. They opted for shaving the excess hair in the face. They used to shave once in two

weeks. 17 subjects in the PCOS group and 13 subjects in the non PCOS group showed reduction in the severity of hirsutism. Probably the others needed an increase in the dosage of cyproterone acetate or duration of treatment. However all of them need to be followed up after 1 year whether there is recurrence or not. 4 subjects were diagnosed as having diabetes mellitus in our study (1 in the PCOD group and 3 in the non PCOD group). 1 subject with PCOD was diagnosed as having impaired glucose tolerance.

In a study by Carmina et al., androgen levels remained low after 1 year of treatment with dexamethasone along with spironolactone[16]. In our study, we gave treatment for 6 months only but did not measure post treatment free testosterone levels. There was statistically significant reduction in the severity of hirsutism in both PCOS and non PCOS groups. We did not follow up the patient after 1 year in our study. In their study, those who were treated with spironolactone only, the hirsutism scores returned to baseline scores after 1 year.

A study by Marescalchi et al., Italy showed that OCP containing EE-CPA was most efficacious in treating hirsutism[17]. In their regimen the dosage of EE is 0.01 mg in first week, 0.02 mg EE/Day for second week and 0.01 mg EE/day in third week with a gap of 7 days, then 12.5 mg CPA/day during first 10 days of every month for 12 months[23]. In our regimen OCP containing EE-CPA in dosage of 35 mcg of ethinyl estradiol and 2 mg of cyproterone acetate for 21 days in a month with a gap of 7 days was given for 6 months. Metformin was given additionally in a dosage of 500 mg BD if BMI is >25 kg/m^2. In their study, the

hirsutism scoring was done after 6, 9 and 12 months. The decrease was -60±18%, -20±11%, 28±21% after 6,9 and 12 months respectively.

In our study the hirsutism scoring was done after 6 months of treatment. The cycles became regular in 46.57% PCOS subjects and 19.17% non PCOS subjects, remained irregular in 4.8% PCOS cases and 4.5% of non PCOS subjects. 4.7% PCOS subects were oligomenorrhoeic and 7.8% non PCOS subjects were amenorrhoeic in our study.

In a study by K Rautio et al., 35 women with PCOS (18 obese and 17 nonobese) were randomised to 6 months treatment with metformin or EE_2-CPA OCP[28]. Metformin treatment had beneficial effects on lipid profile and blood pressure and therefore it could be useful in the prevention of cardiovascular complications in these women[22]. In our study, metformin was used for subjects with BMI>25 kg/m^2, but follow up of the subjects with post treatment lipid profile was not done. 11 of the subjects in our study had abnormal lipid profile (ie, elevated total cholesterol and elevated low density lipoprotein).

There are not many studies in androgen secreting ovarian tumours. In our study, there were 4 cases of androgen secreting ovarian tumours. There was one malignant granulosa cell tumour, one juvenile granulosa cell tumour, two malignant sertoli leydig cell tumours. All the four had features of virilization. Of these, 3 showed marked reduction in severity of hirsutism, but the clitoromegaly, breast atrophy, hoarseness of voice did not change. One case of malignant sertoli leydig cell tumour stage 3 developed recurrence after 6 months and died due to lung metastasis.

There were 6 subjects with hypothyroidism in the non PCOD group. They were given EE-CPA along with thyroxine for six months. 5 subjects showed reduction in the severity of hirsutism by 1 to 3 scores. 1 subject showed no reduction in the severity of hirsutism.

One subject with PCOD showed frontal hair loss. She was obese with BMI of 30.01kg/m^2, had elevated LDL, cholesterol and TGL. She came with complaints of infertility and irregular cycles once in 3 to 6 months. She was given metformin along with EE-CPA for 3 months. She had a hirsutism score of 21. She showed reduction in weight and she had regular cycles after 3 months. She was started on letrozole from the second day of periods but she failed to conceive. She also showed no reduction in the severity of hirsutism.

Obesity has a negative impact on the efficacy of the treatment of hirsutism. Hence appropriate lifestyle advice is necessary for a successful treatment programme[30].

PICTURE NO.7

PICTURE OF A SUBJECT WITH PCOD WITH FRONTAL HAIR LOSS

PICTURE NO.8

PICTURE OF A SUBJECT WITH ANDROGEN SECRETING OVARIAN TUMOUR WITH FRONTAL BALDNESS

She underwent TAH with BSO. Histopathology report came as Juvenile Granulosa Cell Tumour.

Any disturbance in ovarian androgen metabolism will profoundly affect the reproductive state of a woman[35]. Significance of elevated serum LH, insulin resistance or polycystic appearing ovaries assessed by USG for the diagnosis of PCOS remains uncertain. Hyperandrogenism and chronic anovulation are mandatory for the diagnosis of PCOS[36]. Polycystic ovaries are common in normal women[37]. Women who are overweight can expect an improvement in their symptoms if they lose weight[38,39]. Pharmacologic and non pharmacological methods are used for hirsutism. Advances in laser hair removal methods and topical hair growth retardants offer new options[40].

Our study has shown that OCP containing EE-CPA along with metformin has been effective in reducing body weight and regularization of cycles and mild reduction in the severity of hirsutism.

SUMMARY

SUMMARY

In this study, 52 had ovarian causes, (46 – PCOS, 4- ovarian tumour, 2- premature ovarian failure), one had adrenal cause (late onset CAH), systemic cause (hypothyroid -6) for hirsutism.

- Majority of cases of hirsutism in this study fall in the 21 – 30 years age group.
- In addition to hirsutism, menstrual irregularity is the chief complaint
- Majority had hirsutism scoring of 9 to 11. Severe hirsutism (Scoring more than > 15) was seen in androgen secreting ovarian tumour and PCOS (4 cases)
- Virilization features commonly were seen in all the 4 ovarian tumors and late onset CAH . Highly elevated free testosterone levels were seen in androgen secreting ovarian tumours and late onset congenital adrenal hyperplasia.
- Subjects with hirsutism were either overweight or obese except ovarian tumours and CAH.
- Co morbid conditions like hyperlipidemia, diabetes mellitus, hypertension were seen in few PCOS and few non PCOS subjects.
- Level of the testosterone did not correlate with the severity of hirsutism in the PCOS group.

CONCLUSION

CONCLUSION

This study evaluates the various causes of hirsutism, their clinical and biochemical profile and their outcome after treatment. Although PCOS is the most common cause for hirsutism, other causes should also be thought of. The treatment should be individualised depending upon the cause of hirsutism. We should have a high suspicion of virilizing tumours if features of virilization are present. Hirsutism has a response to OCP containing cyproterone acetate but the reduction in severity of hirsutism is mild. They might need increase in the duration of treatment and dosage of cyproterone acetate. Free testosterone and DHEAS may or may not be elevated in PCOS. Majority have normal levels. Free testosterone is highly elevated in androgen secreting ovarian tumours. This study has highlighted the importance of evaluating the other causes of hirsutism such as androgen secreting ovarian tumours and adrenal hyperplasia. Hirsutism causes cosmetic problem and psychological upset on a woman. It can also be a manifestation of an underlying health problem. Hence it should be properly treated so that a favourable outcome can be obtained.

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CLINICAL & BIOCHEMICAL PROFILE OF WOMEN

PRESENTING WITH HIRSUTISM AND ITS TREATMENT

OUTCOME-A PROSPECTIVE STUDY

PROFORMA

Name : Age: IP/OP No:

Presenting complaints:

Hirsutism: Age of onset:

Duration:

Menstrual history:

Duration:

Oligomenorrhoea:

Amenorrhoea:

Marital history:

Duration of infertility:

Recent weight gain:

History of drug intake –specify name of the drug and duration of drug intake

Past history: Previous history of any treatment

Clinical examination Ht Cm BMI Kg/m^2 BP mm Hg

Hirsutism scoring Wt Kg WHR

MODIFIED FERRIMAN -GALLWEY SCORING SYSTEM

Each area is given a score of 1 to 4

1 –mild; 2-moderate; 3- complete light coverage; 4-heavy coverage

Areas

- UPPER LIP
- CHIN
- UPPER BACK

- LOWER BACK
- UPPER ARMS
- THIGHS
- CHEST
- UPPER ABDOMEN
- LOWER ABDOMEN

Score >8 is considered as hirsutism

Signs of virilization and hyperandrogenism

Acne
Hirsutism
Male pattern balding
Increased muscle mass
Deep Voice
Clitoromegaly
Breast atrophy
Acanthosis nigricans
Features of Cushing's syndrome like moon facies, buffalo hump are also looked for. Features of hyperprolactinemia like milk secretion are also seen in all subjects.

INVESTIGATIONS

Serum free TESTOSTERONE

DHEAS

17(OH) PROGESTERONE in subjects with suspected congenital adrenal hyperplasia.

FASTING BLOOD SUGAR
LIPID PROFILE

24 HOUR FREE URINARY CORTISOL (if CUSHING'S SYNDROME is suspected) .

TSH

USG pelvis

USG abdomen if needed

CT abdomen if needed

TREATMENT

- METFORMIN if BMI> 25 kg/m^2
- OCP containing cyproterone acetate 2mg & ethinyl estradiol 35 microgram.
- OVULATION INDUCTION with LETROZOLE

FOLLOW UP IS DONE AFTER 6 MONTHS

- REGULARISATION OF CYCLES is enquired into each subject.
- SEVERITY OF HIRSUTISM is assessed by modified Ferriman- Gallwey scoring system after treatment for 6 months.
- REDUCTION IN WEIGHT is assessed by measuring in Kg & comparing the pretreatment weight.
- CONCEPTION .Pregnancy is confirmed by urine gravindex and ultrasonogram pelvis in 1^{st} trimester.

INFORMED CONSENT FORM

CLINICAL AND BIOCHEMICAL PROFILE OF WOMEN PRESENTING WITH HIRSUTISM AND ITS TREATMENT OUTCOME-A PROSPECTIVE STUDY

Study Centre : Institute of Obstetrics & Gynaecology.
Madras Medical College,Chennai-600 008.

Patient Name :

Patient Age :

IP/ OP NO :

PART – I

Hirsutism in the female means an excessive production of hair with a tendency to male distribution “Excessive” is defined as beyond social acceptability or causing embarrassment to the patient.

Causes: Idiopathic, PCOS, Androgen producing tumours, Congenital Adrenal Hyperplasia, Drugs (Phenytoin, Diazoxide, Minoxidil, Androgen Containing Compounds)

The study is being performed to find the various causes of hirsutism by appropriate clinical and biochemical methods.

I am giving consent for taking blood, urine, ultrasonogram and clinical examination.

I have been informed that I will be given treatment according to the cause as per the hospital guidelines.

After the treatment there will be reduction in the severity of hirsutism, regularization of cycles, conception and/or reduction in weight.

PART – II

Patient may check () these boxes

I confirm that I have read and understood the Information Sheet for the above study. I have the opportunity to ask questions and all my questions and doubts have been answered to my complete satisfaction. I have been informed about the reason for investigating the cause of my hirsutism

I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my legal rights being affected.

I understand that the Clinical study personnel, the Ethics Committee and the Regulatory Authorities will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I agree to take part in the above study and to comply with the instructions given during the study and to faithfully co-operate with the study team, and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.

I hereby consent to participate in this study on Clinical and Biochemical profile of Hirsutism.

I hereby give permission to undergo complete clinical examination diagnostic tests including hematological, biochemical, radiological tests.

Signature/ Thumb Impression: Place Date

Patient’s Name, Address & Ph No:

Name of the witness Witness signature Date

Signature of the investigator: Place Date

Study investigator’s Name:

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

"பெண்களுக்கு ஆண்களைப்போன்று உடம்பில் அதிக முடி வளர்வது - மருத்துவ மற்றும் உயிர்வேதியியல் மாற்றங்கள் மற்றும் சிகிச்சையின் வெளியீடுகள்"

ஆய்வு செய்யப்படும் இடம் : மகப்பேறு அரசு மருத்துவமனை

பங்கு பெறுபவரின் பெயர் : பங்கு பெறுபவரின் பிறந்த தேதி:

பங்கு பெறுபவரின் வயது :

பகுதி -1

நோயாளியின் தகவல் படிவம்

- Hirsutism என்பது பெண்களுக்கு ஆண்களைப் போன்று முடிவளர்தல், அதற்கு பல்வேறு காரணங்கள் உள்ளன.
- சிலருக்கு இயல்பாகவே எந்தக் காரணமும் இன்றி முடி அதிகளவில் வளர்கிறது. அவர்களுக்கு இயக்குநீர் (Hormone) அளவு சாதாரண அளவில் (Normal) உள்ளது.
- சிலருக்கு டெஸ்டோஸ்டிரான் என்ற இயக்குநீர் அதிகமான அளவில் உள்ளதால் முடி வளர்கிறது.
- இந்த இயக்குநீர் பிட்யூட்டரி, அட்ரினல், குரப்பை, கல்லீரல் ஆகியவற்றில் இருந்து சுரக்கிறது.
- இந்த இயக்குநீர் அதிகமாக சுரப்பதால் சிலருக்கு மாதவிலக்கு சீராக வருவதில்லை. சிலருக்கு கர்ப்பம் தரிப்பதில் சிரமம் உள்ளது. சிலருக்கு ஆண்மையான தோற்றத்தை அளிக்கிறது.
- இதன் காரணங்களை கண்டறிந்து அதற்கு ஏற்ப சிகிச்சை அளிப்பதால் இயக்குநீர் சீராக சுரப்பதற்கும், மாதவிலக்கு சீராக வருவதற்கும், கருத்தரிப்பதற்கும், உடலில் முடி வளர்தல் குறைவதற்கும் வாய்ப்பு ஏற்படுகிறது.
- அதன் காரணங்கள் என்ன என்று கண்டறிவதற்காகத்தான் இரத்தம், சிறுநீர், மருத்துவப் பரிசோதனை மற்றும் ஸ்கேன் செய்யப்படுகிறது.

- இந்த ஆய்வினால் எனக்கு எவ்வித பாதிப்பு இல்லை. இந்நோயை எளிதில் கண்டறிய எங்கள் மருத்துவமனை வரைமுறைப்படி சிகிச்சை அளிக்கப்படும்.

பகுதி - 2

பங்கு பெறுவர் இதனை (✓) குறிக்கவும்.

எனக்கு அதிகமாக உடம்பில் முடி வளர்வதற்கான காரணங்கள் கூறப்பட்டது. அந்த காரணங்களை கண்டுபிடிப்பதற்கு இரத்தம், சிறுநீர், ஸ்கேன் பரிசோதனை தேவை என்பதும் கூறப்பட்டது. காரணங்களுக்கு ஏற்ப எனக்கு சிகிச்சை அளிக்கப்படும் எனவும் கூறப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.	☐
நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் இது பொருந்தும் என அறிகிறேன்.	☐
இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளை மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன்.	☐
இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோயக்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.	☐
இந்த ஆய்வில் எனக்கு இரத்தம், ஸ்கேன் பரிசோதனை செய்து கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன்.	☐

பங்கேற்பவரின் கையொப்பம் ..... இடம் ..... தேதி

கட்டை விரல்ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

சாட்சியாளர் பெயர்..... சாட்சியாளர் கையொப்பம் தேதி

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர்.....

ABBREVIATIONS

SHBG	-	Sex hormone binding globulin
PCOS	-	Polycystic Ovarian Syndrome
DHEA	-	Dehydroepiandrosterone
17(OH) Progesterone	-	17 hydroxy Progesterone
DHEAS	-	Dehydroepiandrosterone Sulphate
CAH	-	Congenital Adrenal Hyperplasia
BMI	-	Body Mass Index
CT	-	Computed tomography
USG	-	Ultrasonogram
TSH	-	Thyroid stimulating hormone
CHO	-	Cholesterol
TC	-	Total cholesterol
HDL	-	High density lipoprotein
LDL	-	Low density lipoprotein
VLDL	-	Very low density lipoprotein
EE-CPA	-	Oral contraceptive pill containing cyproterone acetate 2mg and Ethinyl estradiol 35 microogram
COCs	-	Combined oral contraceptive pills
OCP	-	Oral Contraceptive Pill
TRH	-	Thyrotropin releasing hormone
FSH	-	Follicle stimulating hormone
LH	-	Leutinizing hormone

MASTER CHART

SL. NO	NAME	AGE	M/s	*PRIMARY PRESRENTING COMPLAINTS*			DUR . HIRS	HIRS.SCORE	BMI(kg/ m^2)	WHR	OTHERS	FAMILY HISTORY	DRUG INTAKE	USG
				INFERTILITY	*MENSTRUAL*	*WEIGHT GAIN*								
1	SHEELA	20	1	*1*	*2*	*1*	3	11	32.04	0.84	0	NO	-	PCOD
2	ANANDHI	27	1	*1*	*2*	*2*	10	12	24.4	0.82	0	NO	-	PCOD
3	SREEMATHY	22	1	*1*	*1*	*1*	11	13	27.38	0.83	0	NO	-	PCOD
4	MARAGATHM	26	1	*1*	*1*	*1*	7	10	31	0.84	0	NO	-	PCOD
5	PRIYA	24	1	*1*	*1*	*1*	2	14	28.32	0.84	1	NO	-	PCOD
6	MAHA	25	1	*1*	*2*	*1*	7	11	32	0.85	0	YES	-	PCOD
7	SHAFEEN	23	1	*1*	*1*	*1*	10	12	28.88	0.83	1,2,7,9	NO	-	PCOD
8	AISHA	21	1	*1*	*1*	*1*	5	9	30	0.84	1	YES	-	PCOD
9	MALATHY	25	1	*1*	*2*	*1*	11	9	30	0.83	1	YES	-	PCOD
10	NAGAKANI	25	1	*1*	*2*	*1*	5	11	30	0.84	0	NO	-	PCOD
11	ANANDHI	20	1	*1*	*2*	*1*	3	10	28	0.83	0	NO	-	PCOD
12	SUGUNA	30	1	*1*	*1*	*1*	14	11	31	0.84	1	NO	-	PCOD
13	SUMATHY	30	1	*1*	*1*	*1*	3	10	28.8	0.84	2,7	NO	-	PCOD
14	MUNIAMA	30	1	*1*	*1*	*1*	5	14	31	0.85	2	YES	-	PCOD
15	UMA	23	1	*1*	*1*	*1*	4	11	28	0.84	0	NO	-	PCOD
16	JAYANTHY	26	1	*1*	*1*	*1*	2	14	35	0.85	2	YES	-	PCOD
17	HEMAVATHY	26	1	*1*	*2*	*1*	11	13	28	0.81	0	NO	-	PCOD
18	MARYDIANA	29	1	*1*	*1*	*1*	12	12	31	0.82	1	YES	-	PCOD
19	PRAMILA	29	1	*1*	*1*	*1*	5	9	30.66	0.84	1	YES	-	PCOD
20	MEENA	30	1	*1*	*1*	*1*	1	12	36.21	0.9	2,7	YES	_	PCOD
21	MAMATA	21	1	*1*	*1*	*1*	3	13	30	0.8	1	NO	_	PCOD
22	SUMATHY	28	1	*1*	*1*	*1*	8	21	30.01	0.88	2,3	NO	_	PCOD

SL.NO	NAME	FBS mg/dl	*LIPID PROFILE*	*FREE TESTOSTERONE (pg/ml)*	DHEAS(ng/ml)	17(OH)P ng/ml	TSH(mU/ml)	TREATMENT	OUTCOME			
									WT REDUCTION	MENSTRUL CYCLES	HIRSCORE	CONCEPTION
1	SHEELA	62	*NORMAL*	*2 pg/ml*	4.5	_	NORMAL	K,M×3&L,M×3	YES	1	8	-
2	ANANDHI	68	*NORMAL*	*1*	8	_	NORMAL	K,M×3&L,M×3	NO	1	11	CONCEIVED
3	SREEMATHY	88	*NORMAL*	*2.52*	8.5	_	NORMAL	K,M×3&L,M×3	YES	1	13	-
4	MARAGATHM	75	*NORMAL*	*3.09*	11	_	NORMAL	K,M×3&L,M×3	YES	1	10	-
5	PRIYA	84	*NORMAL*	*1.91*	8	_	NORMAL	K,M×3&L,M×3	YES	1	10	CONCEIVED
6	MAHA	89	*NORMAL*	*2*	8	_	NORMAL	K,M×3&L,M×3	YES	1	11	-
7	SHAFEEN	168	*ABNORMAL*	*4.2*	11	_	NORMAL	DEFAULTED				
8	AISHA	108	*NORMAL*	*4.25*	12	_	NORMAL	DEFAULTED				
9	MALATHY	94	*NORMAL*	*1*	5	_	NORMAL	K,M×3&L,M×3	YES	1	8	CONCEIVED
10	NAGAKANI	84	*NORMAL*	*2.61*	8	_	NORMAL	K,M×3&L,M×3	YES	1	10	CONCEIVED
11	ANANDHI	86	*NORMAL*	*1*	7	_	NORMAL	K,M×3&L,M×3	YES	1	8	CONCEIVED
12	SUGUNA	84	*NORMAL*	*2*	8	_	NORMAL	K,M×3&L,M×3	YES	2	11	-
13	SUMATHY	72	*NORMAL*	*4*	11	_	NORMAL	K,M×3&L,M×3	YES	1	10	-
14	MUNIAMA	89	*NORMAL*	*1.4*	7	_	NORMAL	K,M×3&L,M×3	YES	1	14	-
15	UMA	88	*NORMAL*	*2*	10.4	_	NORMAL	K,M×3&L,M×3	YES	1	9	-
16	JAYANTHY	90	*ABNORMAL*	*1.2*	10.4	_	NORMAL	K,M×3&L,M×3	YES	1	14	-
17	HEMAVATHY	92	*NORMAL*	*0.7*	8.2	_	NORMAL	K,M×3&L,M×3	YES	1	8	CONCEIVED
18	MARYDIANA	94	*NORMAL*	*1*	10.5	_	NORMAL	K,M×3&L,M×3	YES	1	12	-
19	PRAMILA	91	*NORMAL*	*5.8*	6.8	_	NORMAL	K,M×3&L,M×3	YES	1	9	-
20	MEENA	130	*ABNORMAL*	*1.2*	10.4	_	NORMAL	K,M×3&L,M×3	YES	1	12	-
21	MAMATA	82	*NORMAL*	*0.9*	8	_	NORMAL	K,M×3&L,M×3	YES	1	13	
22	SUMATHY	88	*ABNORMAL*	*2*	8	_	NORMAL	K,M×3&L,M×3	YES	1	21	

SL.NO	NAME	AGE	M/s	*PRIMARY PRESRENTING COMPLAINTS*			DUR . HIRS	HIRS.SCORE	BMI(Kg/ m^2)	WHR	OTHERS	FAMILY HISTORY	DRUG INTAKE	USG
				INFERTILITY	MENSTRUAL	WEIGHT GAIN								
23	REKHA	19	2	*2*	1	1	7	9	30	.84	1	YES	-	PCOD
24	SUVAHANI	21	2	*2*	1	1	11	9	29.06	0.84	0	NO	_	PCOD
25	SUMATHY	19	2	*2*	1	1	7	11	32	0.84	0	YES	_	PCOD
26	NIRMALA	21	2	*2*	1	1	4	11	25	0.8	1	NO	_	PCOD
27	DURGA	23	2	*2*	1	1	5	11	34.2	0.86	1	NO	_	PCOD
28	THARA	25	1	*2*	1	1	6	12	30	0.84	1	YES	_	PCOD
29	SARADHA	30	2	*2*	1	1	6	16	28	0.82	1	NO	_	PCOD
30	STELLAMARY	30	1	*2*	2	1	3	13	33.71	0.87	2	NO	_	PCOD
31	GEETHA	32	1	*2*	1	1	6	11	29	0.83	0	NO	_	PCOD
32	HEMAMALINI	20	2	*2*	1	1	3	9	28	0.8	0	NO	_	PCOD
33	PRABAVATHY	22	2	*2*	1	1	7	17	32	0.84	1,2	YES	_	PCOD
34	SINDHUJA	22	2	*2*	1	2	2	13	20.54	0.8	1	NO	_	PCOD
35	PRIYA	20	2	*2*	1	1	1	20	25.39	0.8	1	NO	_	PCOD
36	SONIA	25	2	*2*	1	1	1	9	30	0.82	0	NO	_	PCOD
37	JENITA	23	2	*2*	1	1	1	9	26	0.8	0	NO	_	PCOD
38	PRIYA	32	2	*2*	2	1	1	9	30	0.84	0	NO	_	PCOD
39	SAHANA	16	2	*2*	1	1	1	11	29	0.83	1	NO	_	PCOD
40	PRIYA	19	2	*2*	1	1	1	12	26	0.82	1	NO	_	PCOD
41	JENIFER	18	2	*2*	1	1	2	10	26	0.83	8	NO	_	PCOD
42	KAVITHA	23	2	*2*	1	1	1	9	30	0.84	1	NO	_	PCOD
43	VIJI	20	2	*2*	1	1	2	11	28	0.82	0	NO	-	PCOD

SL.NO	NAME	FBS mg/dl	LIPID PROFILE	FREE TESTOSTERONE (pg/ml)	DHEAS(ng/ml)	17(OH)P ng/ml	TSH(mU/ml)	TREATMENT	OUTCOME			
									WT REDUCTION	MENSTRUL CYCLES	HIRS SCORE	CONCEPTION
23	REKHA	82	NORMAL	1.2	7.5	-	NORMAL	K+Mx6	YES	1	7	-
24	SUVAHANI	92	NORMAL	1.5	7	_	NORMAL	K+M×6	YES	1	9	-
25	SUMATHY	94	NORMAL	0.8	9.6	_	NORMAL	K+M×6	YES	1	8	-
26	NIRMALA	88	NORMAL	1	10.5	_	NORMAL	K×6	NO	1	11	-
27	DURGA	86	NORMAL	2	7.5	_	NORMAL	K+M×6	YES	3	11	-
28	THARA	86	NORMAL	1.2	8	_	NORMAL	K+M×6	YES	1	8	-
29	SARADHA	88	NORMAL	1.9	8	_	NORMAL	K+M×6	YES	3	16	-
30	STELLAMARY	92	NORMAL	4.6	14.3	_	NORMAL	K+M×6	YES	1	11	-
31	GEETHA	84	NORMAL	0.9	8	_	NORMAL	K+M×6	NO	2	11	-
32	HEMAMALINI	86	NORMAL	0.9	7	_	NORMAL	K+M×6	YES	1	9	-
33	PRABAVATHY	110	ABNORMAL	2	10.5	_	NORMAL	K+M×6	YES	2	17	-
34	SINDHUJA	90	NORMAL	0.9	4.6	_	NORMAL	K×6	YES	1	11	-
35	PRIYA	92	NORMAL	0.8	10	_	NORMAL	K+M×6	YES	1	20	-
36	SONIA	86	NORMAL	1	7	_	NORMAL	K+M×6	YES	1	7	--
37	JENITA	74	NORMAL	1	6.8	_	NORMAL	K+M×6	YES	1	7	-
38	PRIYA	82	NORMAL	1.5	7.5	_	NORMAL	K+M×6	YES	1	9	-
39	SAHANA	90	NORMAL	1	8.5	_	NORMAL	K+M×6	YES	1	12	-
40	PRIYA	75	NORMAL	1.2	8	_	NORMAL	K+M×6	YES	1	12	-
41	JENIFER	82	NORMAL	1.5	8	_	ELEVATED	K+M+T×6	YES	1	10	-
42	KAVITHA	94	NORMAL	1.8	9	_	NORMAL	K+M×6	YES	3	9	--
43	VIJI	84	NORMAL	1.8	9	_	NORMAL	DEFAULTED				

SL.NO	NAME	AGE	M/s	PRIMARY PRESRENTING COMPLAINTS			DUR. HIRS	HIRS.SCORE	BMI(kg/ /m^2)	WHR	OTHERS	FAMILY HISTORY	DRUG INTAKE	USG
				INFERTILITY	MENSTRUAL	WEIGHT GAIN								
44	MINI	28	2	2	2	1	1	11	30	0.84	0	NO	_	PCOD
45	KEERTIKA	21	2	2	2	1	1	9	29	0.83	0	NO	_	PCOD
46	MURUGAM	20	2	2	1	1	3	10	28	0.82	1	NO	_	PCOD
47	MALLIGA	31	1	2	2	2	10	9	21.48	0.8	0	YES	_	NORMAL
48	ARUL	26	1	1	1	1	6	13	32	0.88	2	NO	_	NORMAL
49	NITHYA	30	1	1	2	2	3	11	19.3	0.79	0	YES	_	NORMAL
50	SUMATHY	30	1	1	2	1	10	12	30	0.84	0	NO	_	NORMAL
51	PONNI	24	2	2	2	1	2	11	22	0.8	0	NO	_	NORMAL
52	VENI	28	2	2	2	2	11	12	24	0.8	0	YES	_	NORMAL
53	RANI	27	2	2	2	1	11	12	24.88	0.8	0	NO	_	NORMAL
54	SUJATHA	28	2	2	1	1	10	11	28	0.82	0	YES	_	NORMAL
55	UMARANI	39	2	2	1	2	12	15	20	0.79	9	NO	ART for 4 years	NORMAL
56	CHITRA	40	2	2	2	2	15	10	30	0.85	7	YES	_	NORMAL
57	MALLIGA	40	1	2	1	2	4	12	28	0.8	3,5	NO	_	ADNEXAL MASS
58	ELIZABET	28	1	2	1	1	1	9	25	0.8	0	NO	_	NORMAL
59	GEETHA	40	1	1	1	1	1	9	28	0.84	1	NO	DANAZOLx2 years	ENDOMETRIOSIS
60	SARITHA	26	1	2	1	2	2	9	29	0.85	3,5	NO	CHLORPROMAZINEx5 years	ADNEXAL MASS
61	KALA	43	1	2	1	1	2	10	25.39	0.86	7,9	NO	_	ADNEXAL MASS
62	SHEELA	30	1	2	2	1	5	12	36.21	0.9	0	NO	_	NORMAL
63	GOVINDAM	51	1	2	2	1	25	9	30	0.85	7,9	YES	_	NORMAL
64	NAGAMA	50	1	2	1	2	1	20	20	0.76	3,4,5	NO	_	ADNEXAL MASS

SL.NO	NAME	FBS mg/dl	LIPID PROFILE	FREE TESTOSTERONE (pg/ml)	DHEAS(ng/ML	17(OH)P ng/ml	TSH(mU/ml)	TREATMENT	OUTCOME			
									WT REDUCTION	MENSTRUL CYCLES	HIRS SCORE	CONCEPTION
44	MINI	80	NORMAL	1.8	8	_	NORMAL	DEFAULTED				
45	KEERTIKA	86	NORMAL	2	10.5	_	ELEVATED	K+M+Tx6	YES	1	9	
46	MURUGAM	84	NORMAL	1.9	9	_	NORMAL	K+MX6	YES	1	7	
47	MALLIGA	82	NORMAL	1	8	_	NORMAL	Kx3+Lx3	NO	1	9	CONCEPTION
48	ARUL	120	ABNORMAL	1.8	10	_	NORMAL	K+M×3+L+M×3	YES	1	13	
49	NITHYA	102	NORMAL	1	8	_	NORMAL	Kx3+Lx3	NO	1	11	CONCEPTION
50	SUMATHY	100	NORMAL	1	7.5	_	NORMAL	K+M×3+L+M×3	YES	1	12	
51	PONNI	94	NORMAL	1.9	10.5	_	NORMAL	KX6	NO	1	11	
52	VENI	88	NORMAL	1.4	3.8	_	NORMAL	KX6	NO	1	12	
53	RANI	86	NORMAL	1.2	7	_	NORMAL	KX6	NO	1	12	
54	SUJATHA	84	NORMAL	1	9	_	NORMAL	POF→HRT	NO	4	10	
55	UMARANI	160	ABNORMAL	4.2	11	_	NORMAL	POF→HRT	NO	4	12	
56	CHITRA	158	ABNORMAL	4.6	8	_	NORMAL	DEFAULTED				
57	MALLIGA	94	NORMAL	3.9	10.4	_	NORMAL	TAH WITH BSO	NO	3	10	
58	ELIZABET	88	ABNORMAL	4	7.5	_	ELEVATED	K+T×6	NO	1	9	
59	GEETHA	86	NORMAL	0.8	9.6	_	NORMAL	PATIENT WENT FOR IVF				
60	SARITHA	78	NORMAL	1.2	7.5	_	NORMAL	TAH WITH BSO	NO	3	7	
61	KALA	148	ABNORMAL	0.8	8.5	_	NORMAL	TAH WITH BSO	TOPICAL TREATMENT			
62	SHEELA	98	ABNORMAL	1.2	10.4	_	NORMAL	K+M×6	YES	1	10	
63	GOVINDAM	160	ABNORMAL	0.8	7.5	_	NORMAL	TOPICAL TREATMENT				
64	NAGAMA	92	NORMAL	5.8	8	_	NORMAL	TAH WITH BSO	YES	3	15	

SL. NO	NAME	AGE	M/s	PRIMARY PRESRENTING COMPLAINTS			DUR. HIRS	HIRS.SCORE	BMI(kg/m^2)	WHR	OTHERS	FAMILY HISTORY	DRUG INTAKE	USG
				INFERTILITY	MENSTRUAL	WEIGHT GAIN								
65	GOMATHY	30	2	2	2	2	2	10	28	.83	0	NO	-	NORMAL
66	ANITHA	22	2	2	2	1	3	11	29	0.84	0	NO	_	NORMAL
67	SREEMATHY	25	2	2	1	1	2	10	28	0.82	1	NO	_	NORMAL
68	SELVI	22	2	2	1	1	1	10	30	0.85	1	NO	_	NORMAL
69	DAISY	18	2	2	1	1	1	9	29	0.84	1	NO	_	NORMAL
70	KALIAMA	22	2	2	1	1	1	11	26	0.83	0	NO	_	NORMAL
71	GURUVAMA	30	2	2	1	1	2	12	28	0.82	0	NO	_	NORMAL
72	PRIYA	15	2	2	1	2	5	14	22	0.79	5,6	NO	_	ADRENAL HYPERPLASIA with HYPOPLASTIC UTERUS
73	RAMAJEYAM	19	2	2	1	2	1	16	19.3	0.76	4,5,6	NO	_-	ADNEXAL MASS

SL.NO	NAME	FBS mg/dl	LIPID PROFILE	FREE TESTOSTERONE (pg/ml)	DHEAS(ng/ml)	17(OH)P ng/ml	TSH(mU/ml)	TREATMENT	OUTCOME			
									WT REDUCTION	MENSTRUL CYCLES	HIRS SCORE	CONCEPTION
65	GOMATHY	88	NORMAL	1	8	-	NORMAL	Kx6	YES	1	10	
66	ANITHA	86	NORMAL	1.5	8.2	_	ELEVATED	K+M+T×6	YES	1	9	
67	SREEMATHY	84	NORMAL	1	8.5	_	ELEVATED	K+M+T×6	YES	1	8	
68	SELVI	82	NORMAL	1	7	_	ELEVATED	K+M+T×6	YES	1	7	
69	DAISY	80	NORMAL	0.8	7.5	_	ELEVATED	K+M+T×6	YES	1	7	
70	KALIAMA	84	NORMAL	0.9	8.5	_	ELEVATED	K+M+T×6	YES	1	9	
71	GURUVAMA	78	NORMAL	1.9	8.5	–	NORMAL	K+M×6	YES	1	12	
72	PRIYA	75	NORMAL	9.58	13.15	14.5	NORMAL	DEXA .5mg OD×6	NO	2	10	
73	RAMAJEYAM	72	NORMAL	3.5	15	–	NORMAL	TAH WITH BSO	RECURRENCE&PATIENT DIED			

KEY TO MASTER CHART

Sl.No	-	Serial number
M/s	-	Marital status
1	-	Married
2	-	Unmarried
Infertility		
1	-	Infertility is present
2	-	No infertility
Menstrual	-	Menstrual complaints
1	-	Regular cycles
2-		Irregular cycles or oligomenorrhoea or amenorrhoea
Weight gain		
1	-	Weight gain
2	-	No weight gain or loss
DUR.HIRS	-	Duration of hirsutism
HIRS.SCORE	-	Hirsutism score
BMI	-	Body Mass Index
WHR	-	Waist hip ratio
OTHERS	-	Include masculinizing features , hypertension and diabetes mellitus
1	-	Acne
2	-	Acanthosis Nigricans
3	-	Frontal hair loss
4	-	Breast Atrophy
5	-	Clitoromegaly

6	-	Hoarseness of voice
7	-	Hypertension
8	-	Generalised hair loss
9	-	Diabetes Mellitus
USG	-	Ultrasonogram
PCOD	-	Polycystic Ovarian disease
FBS	-	Fasting blood sugar
TSH	-	Thyroid stimulating homone
K	-	OCP containing cyproterone acetate 2mg and Ethinyl estradiol 35 mcg
M	-	Metformin
L	-	Letrozole
T	-	Thyroxine
17 (OH)P	-	17 hydroxy progesterone
DHEAS	-	Dehydroepiandrsterone sulphate
POF	-	Premature ovarian failure
HRT	-	Hormone replacement therapy
TAH WITH BSO	-	Total abdominal hysterectomy with bilateral salpingo oophorectomy
IVF	-	In vitro fertilization
ART	-	Antiretroviral therapy